PESTS NOT KNOWN TO OCCUR IN THE UNITED STATES OR OF LIMITED DISTRIBUTION, NO. 58: KARNAL BUNT

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| Disease | KARNAL BUNT |
| Pathogen | Tilletia indica (Mitra) Mundkur |
| Selected Synonyms | Neovossia indica Mitra<br>Partial bunt, Indian bunt |
| Class Order: Family | Basidiomycetes: Ustilaginales: Tilletiaceae |

**Economic Importance**

The economic losses caused by Karnal bunt are manifold. While overall yield losses are usually light, the disease can assume epiphytotic proportions in localized areas. Surveys in northern India have found that, even in years of heavy disease, the total yield loss was only 0.3-0.5 percent (Joshi, Singh, and others 1983, Munjal 1975). In individual fields, however, as much as 89 percent of the kernels were infected with yield losses reaching 20 percent. Severe bunt infection reduced seed germination by over 40 percent (Rai and Singh 1978, Singh 1980). Moreover, the infected seeds that did germinate produced abnormal weak seedlings about 20 percent of the time. Karnal bunt adversely affects the color, odor, and palatability of flour and baked products. A wheat lot with only 3.0 percent Karnal bunt infection was considered unfit for human consumption (Mehdi, Joshi, and Abrol 1973). The presence of Karnal bunt could also severely limit the certified seed and grain exports of any area in which the disease was established.

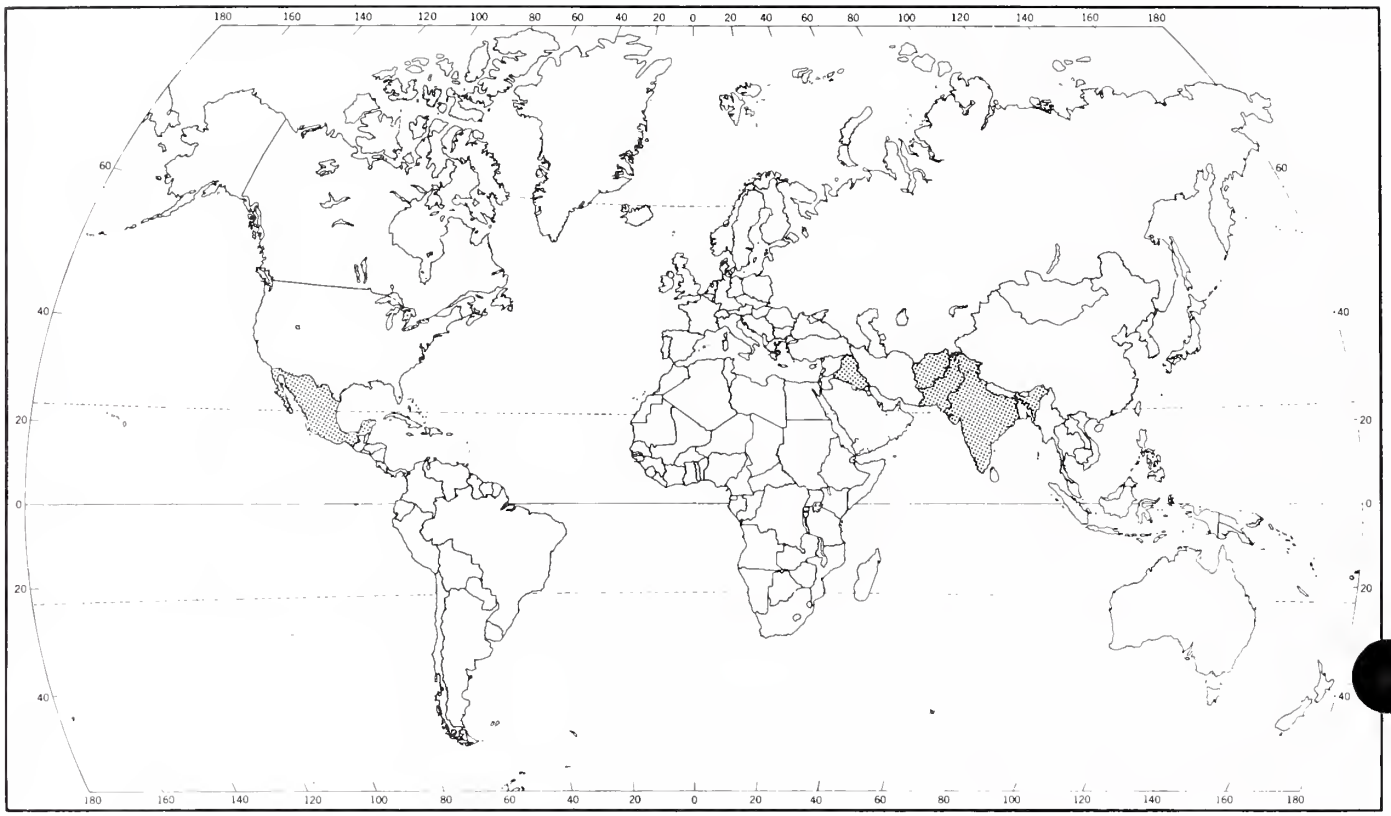
**Hosts**

The most common host of Tilletia indica is Triticum aestivum (bread wheat). It also infects Triticum durum (durum wheat) and X Triticosecale sp. (triticale).

**General Distribution**

Karnal bunt is known to occur in the following countries: Afghanistan, India, Iraq, Mexico, and Pakistan.

The pathogen has also been discovered in exported seed samples from Lebanon and Syria. No other information is available from these countries (Joshi, Singh, and others 1983, Commonwealth Mycological Institute 1974).



Tilletia indica distribution map prepared by Non-Regional Administrative Operations Office and Biological Assessment Support Staff, PPQ, APHIS, USDA

Characters

Teliospores brown to dark brown, spherical or oval, and 24-47 µm in diameter. Exospore adorned with thick, truncate, compact projections. Teliospores germinate into a promycelium which produces sickle-shaped or filiform sporidia, 64-97 µm X 1.6-1.8 µm, at its apex (Duran and Fisher 1961).

The fungus can be cultured by placing fresh sporidia on a variety of media including potato dextrose agar plus 3 percent malt extract, or dextrose-yeast-extract media. Such cultures yield monokaryotic branching mycelia and numerous secondary sporidia (Krisha and Singh 1983, Joshi, Singh, and others 1983).

Characteristic Damage

Tilletia indica does not attack the whole plant systemically but infects each spikelet or floret separately. Localized systemic infection may occur, however, involving florets adjacent to the sites of initial infection (Dhaliwal,

Randhawa, and others 1983). The disease pattern on the host plant is irregular (Fig. 1). Not all spikelets in a head will be infected, and not all grains in a given spikelet become diseased (Bedi, Sikka, and Mundkur 1949).

(Fig. 1)

Tilletia indica on wheat spikes showing darkened kernels, as indicated by arrows (Courtesy California Department of Food and Agriculture).

The disease becomes apparent after the grain has matured. Then the glumes may fall off, exposing the bunted kernels. Diseased kernels are usually only partially destroyed by Karnal

bunt. Affected seed tissue is replaced by a black powdery mass of spores (the sorus). In the case of a mild attack, sporulation is confined to the endosperm along the longitudinal groove of the kernel. In a severe infection, the embryo is also affected (Figs. 2 and 3).

(Fig. 2)

Tilletia indica on wheat showing infection of embryo end and longitudinal groove (USDA photo).

Diseased kernels may be cracked and broken with tissue from the embryo end eroded. Bunted kernels have a fishy odor due to the presence of trimethylamine compounds.

Three other diseases that can be confused with Karnal bunt are black point, common bunt, and dwarf bunt of wheat. These diseases are established in the United States.

Black point or kernel smudge is a disease caused by a number of fungi including Alternaria spp. and Helminthosporium spp. This disease causes the embryo end of the wheat kernel to appear blackened or smudged and sometimes shriveled. The basal groove may also be discolored. There will be no spore-filled sorus as in a bunted kernel and no fetid odor. The thick-walled teliospores of the Karnal bunt fungus are easily distinguished from spores of black point fungi by microscopic examination (Wiese 1977).

(Fig. 3)

Tilletia indica on wheat kernels showing varying degrees of infection. Discolored areas on kernels contain numerous teliospores (USDA photo).

Common bunt (stinking smut) is caused by either of two fungi, Tilletia caries (D.C.) Tul. and T. foetida (Wall.) Liro. Dwarf bunt is caused by T. controversa Kuhn. These diseases infect the wheat seed or seedling and develop systemically in the growing plant. Typically, the entire wheat head is infected in common and dwarf bunt, unlike Karnal bunt where only a few kernels per spike are diseased. In common and dwarf bunt, the entire kernel is converted into a fetid spore-filled sorus. The Karnal bunt sorus only partially replaces the seed (Wiese 1977).

Detection Notes

1. Inoculum in the form of teliospores can enter the country as a surface contaminant on host plants and plant parts. Title 7, Part 319.59 of the Code of Federal Regulations prohibits entry into the United States of seeds, plants, unprocessed straw, chaff, and products of the milling process (other than flour) of Triticum spp. (wheat) from countries where Karnal bunt is known to occur. These commodities can only enter with a USDA departmental permit for scientific purposes. Germplasm samples from experimental breeding stations in Mexico could also provide a means of entry for the disease. In addition, railcars which had been used for wheat transport are sometimes contaminated and must be cleaned before entering the United States from Mexico.

2. Look for bunted seed that is fragile, darkened, and fishy smelling. The kernel usually remains whole although part of the germ end may be eroded. Cracks in the surface reveal the black powdery spore mass within the endosperm at the embryo end of the kernel or along the kernel groove.

3. The disease is difficult to detect in the field unless infection is severe. A laboratory assay using a centrifuge-wash technique is recommended for the detection of teliospores in wheat samples and railway car sweepings (Matsumoto, Boratynski, and others 1984).

4. Kernels suspected of being infected with Tilletia indica should be submitted for identification in a double container (one container within another) with a screw lid.

## Biology and Etiology

Teliospores are the overwintering propagules of Tilletia indica. Fresh teliospores are often incapable of natural germination without a dormancy period of at least 4-9 months. Thereafter, germination is erratic and irregular with some spores remaining viable for up to 4-5 years.

Teliospores capable of producing infection germinate at the soil surface by forming a short promycelium. As many as 65-185 primary sporidia (infectious spores) form at the apex of the promycelium. Large numbers of secondary sporidia can be produced by budding from the primary sporidia or from mycelia (Krishna and Singh 1983, Joshi, Singh, and others 1983).

Sporidia are carried to the wheat spike by air currents and splashing water. Infection occurs only during wheat heading and floral development. Bagging of wheat heads at this time completely blocked infection (Bedi, Sikka, and Mundkur 1949). In contrast, artificial spike inoculation led to a high incidence of disease (Joshi, Singh, and others 1983).

Germ tubes arising from the sporidia directly penetrate the epidermal cells of the glumes and enter the ovary through the ovary wall (Munjal and Chatrath 1976). Mycelia may grow into the endosperm and sometimes into the embryo of the wheat kernel. The infected endosperm is consumed and replaced by large numbers of teliospores. At harvest, teliospores are released to contaminate the soil or wheat seed externally.

Environmental conditions directly affect the epidemiology of the disease. Frequent rains and high relative humidity (70 percent or greater) at the time of heading favor disease development (Bedi, Sikka, and Mundkur 1949, Khetarpal, Agarwal, and Chauhan 1980). Irrigation induced a higher incidence of disease by increasing humidity, thus increasing the length of the wet period, conditions favorable for teliospore germination and disease development (Bedi, Sikka, and Mundkur 1949). Favorable day temperatures of 18-22.5° C and soil temperatures of 17-21° C at ear emergence also increased the severity of Karnal bunt (Aujla, Sharma, and others 1977).

## Control

Karnal bunt is difficult to manage because of the biology of the pathogen and the susceptibility of the host. Teliospores can remain viable for 4-5 years in the soil, making crop rotation impractical. Chemical seed treatment may reduce infection by controlling seedborne inoculum, but it does little to eliminate soilborne inoculum. Use of resistant varieties would be the most effective means of control, but no commercial cultivars are known to be resistant.

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